ABSTRACT OF THE DISCLOSURE

Inhibins and activins are protein hormones that reciprocally modulate a diversity of regulatory pathways. Competitive binding experiments revealed that betaglycan, the type III TGF-β receptor, also functions as an inhibin receptor. Betaglycan augments the binding of inhibin to the ActRII activin receptor. By augmenting inhibin binding to ActRII, betaglycan effectively sequesters ActRII away from activin and thereby reduces activin signaling. In addition, the ActRII-betaglycan complex may generate novel signals distinct from those initiated by activin signaling via ActRII and ALK4. Betaglycan is produced in discrete nuclei of the rat brain and by specific cell types within the adult rat pituitary, testis, and ovary. The presence of betaglycan within inhibin-responsive tissues and cell types, together with the ability of this protoglycan to bind inhibin and to confer inhibin sensitivity, is consistent with a role of betaglycan as an inhibin-specific receptor mediating inhibin responses within various tissues.